Available online at www.sciencedirect.com
ScienceDirect
Drug and Alcohol Dependence 99 (2009) 350–353
DRUG and ALCOHOL DEPENDENCE
www.elsevier.com/locate/drugalcdep

Short communication

Delay discounting by the children of smokers and nonsmokers

Brady Reynolds *, Kristen Leraas, Christine Collins, Shane Melanko

Research Institute at Nationwide Children's Hospital & Department of Pediatrics, The Ohio State University, 700 Children's Drive, J1401 Columbus, OH, USA

Received 24 April 2008; received in revised form 23 July 2008; accepted 29 July 2008
Available online 24 September 2008

Abstract

This research compared delay discounting in mothers and their children (12 or 13 years of age). Half of the mothers ($n = 15$) were current smokers, and the other half ($n = 15$) reported never smoking. Considerable research has shown that adult smokers discount more by delay than nonsmokers, and that parent smoking is a risk factor for adolescent smoking. Thus, it was hypothesized that the mothers who smoked would discount more by delay than the mothers who had never smoked. Also, it was expected that children at increased risk for smoking (i.e., mother is smoker) would discount more by delay than children at lower risk for smoking (i.e., mother is nonsmoker). The results confirmed these hypotheses: mothers who smoked discounted significantly more than nonsmoking mothers; and, in a parallel fashion, children with mothers who smoked discounted significantly more than children of nonsmokers. These findings indicate that delay discounting may be a behavioral risk factor for adolescent cigarette smoking that predates any substantial use of nicotine.

Keywords: Impulsivity; Delay discounting; Child; Smoking; Tobacco; Human

1. Introduction

Delay discounting refers to observations that outcomes decrease in value, or in their effectiveness to exert control over behavior, when delayed. More extreme tendencies to discount value by delay are taken to reflect impulsive behavior (e.g., Green and Myerson, 2004). Consistent with this interpretation, addicted populations (including cigarette smokers) often discount more by delay than matched, non-addicted control participants (see Bickel and Marsch, 2001; Reynolds, 2006, for reviews).

A potential area of delay discounting research that has not been explored is discounting by children or young adolescents who are at risk for addiction. Such research may help define the developmental relationship between delay discounting and drug dependence. For example, if high rates of delay discounting (i.e., more impulsive discounting) are a risk factor for addictive behaviors, then children at risk for addiction should be expected to discount more by delay than children at low risk, thus paralleling findings between adult addicted and non-addicted populations (Bickel and Marsch, 2001; Reynolds, 2006). One study has shown that adult women with no history of alcoholism but who had alcoholic fathers (a risk factor for alcoholism) discounted more by delay than women with no paternal history of alcoholism (Petry et al., 2002). Interestingly, this finding did not extend to men with and without alcoholic fathers. Alternatively, if the high rates of delay discounting that are often associated with drug addiction are a consequence of the drug use itself, then high and low risk children who have not used drugs may not differ in terms of delay discounting.

For the current study, delay discounting was assessed in mothers who smoked and did not smoke cigarettes and also in their biological children. A substantial body of evidence indicates that parent smoking is a risk factor for adolescent smoking (summarized in Tyas and Pederson, 1998), and also that many adolescent smokers go on to become nicotine dependant adult smokers (e.g., Jefferis et al., 2003). Further, the relationship between parental and adolescent cigarette smoking appears to be largely accounted for by the genetic relatedness between the parents and their children (e.g., Maes et al., 2006). For these reasons, we hypothesized for the current study that mothers who smoked would discount more by delay than

* Corresponding author at: Research Institute at Nationwide Children's Hospital & Departments of Pediatrics and Psychology, The Ohio State University, 700 Children's Drive, J1401 Columbus, OH 43205, USA. Tel.: +1 614 722 3549; fax: +1 614 722 3544.
E-mail address: Reynolds.421@osu.edu (B. Reynolds).

0376-8716/$ – see front matter © 2008 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.drugalcdep.2008.07.015

nonsmoking mothers. We also expected this group difference to extend to the children of mothers who smoked and did not smoke.

2. Methods

2.1. Participants

A community sample of mothers and their children (12 or 13 years of age) was recruited from the central Ohio area through posters, advertisements in local newspapers, and word of mouth referrals ($N = 60$). Half of the mothers ($n = 15$) reported currently smoking at least 15 cigarettes per day, and the other half ($n = 15$) reported never smoking (i.e., no lifetime history of smoking cigarettes). Of the mothers who smoked, 73% reported smoking during their pregnancy with the child participant. The children all reported nonsmoking, though three of the children of the mothers who smoked reported having experimented with cigarettes. These three reports of past experimentation were the only reports of smoking for the children. All statistical analyses were conducted with and without these three experimenters included, and the results were unchanged by their inclusion or exclusion. Therefore, all future reference to the children of mothers who smoked will include these children who had experimented with smoking. For this study, we did not attempt to collect data about the biological fathers of the child participants.

2.2. Procedure

All data collection took place in a human-behavior laboratory at the Research Institute at Nationwide Children's Hospital, Department of Pediatrics, The Ohio State University. All participants reviewed and signed consent and assent forms approved by the Research Institute IRB. Participation involved a single laboratory session for each mother and child dyad. Mothers and their children provided samples of breath (for carbon monoxide) and urine (for cotinine) to verify cigarette smoking status.

Delay discounting was assessed for all participants with the Kirby Delay Discounting Questionnaire (Kirby et al., 1999). With this measure, delay discounting is determined from 27 hypothetical choice questions for either immediate or delayed money, with the delays ranging from 7 to 186 days. While the choices are hypothetical, participants are instructed to make choices as though they are actually going to receive the money they choose. The Kirby was scored as described previously by Kirby et al. (1999), with k values (an index of delay discounting) assigned to participants along a range of 10 discreet steps: .00016, .00025, .00063, .0016, .0039, .010, .0126, .065, .16, and .25. This measure provides three separate k values for each participant for large ($85, $80, and $75), medium ($60, $55, and $50), and small ($35, $30, and $25) delayed monetary amounts. Values of k were assigned according to choice patterns across the 27 items, with more choices for smaller immediate amounts associated with larger k values. Larger k values indicate greater delay discounting of value for the delayed options.

2.3. Analyses

Using SPSS Version 15©, tests of normality (Shapiro-Wilk) were performed on all k values. None of the delay discounting data were normally distributed; therefore all k values were Log 10 transformed. With these transformed values, a between-subjects ANOVA was performed to compare average k values between mothers who smoked and did not smoke. For the children, a between-subjects two-way ANOVA (smoking status and gender) was performed to compare average k values across those with and without mothers who smoked. Significant differences in the ANOVA models were explored further with independent samples t-tests to examine group differences across the three different reward magnitudes of the Kirby assessments. For these analyses, there was a Bonferroni correction (significant $p \leq .016$) to reduce the likelihood of Type I error resulting from these multiple statistical tests. A Pearson's correlation test was used to explore the association between k values for mothers and their children; while partial eta squares (η_p^2) were used to estimate effect sizes for all group comparisons.

3. Results

Participant demographic information is presented in Table 1. For the mothers, smokers had higher carbon monoxide and cotinine levels than nonsmokers, thus confirming smoking status. The two groups also differed on race and IQ. The children also differed on race and IQ, but they were similar on the biomarkers of smoking—which were consistent with nonsmoking. However, the children of nonsmoking mothers reported more caffeine intake.

For mothers, there was a significant effect of smoking status on delay discounting, with mothers who smoked discounting more than mothers who did not smoke [$F(29, 1) = 6.31$, $p = .016$; $\eta_p^2 = .197$]. This difference was accounted for by group differences in k values for medium [$t(28) = 2.93$, $p = .007$; $\eta_p^2 = .241$] and large [$t(28) = 2.66$, $p = .013$; $\eta_p^2 = .207$] reward magnitudes (see top panel of Fig. 1). For the children, there was no significant interaction between smoking status and gender on k values, and there was no main effect of gender. However, there was an effect of mother smoking status [$F(29, 1) = 8.01$, $p = .006$; $\eta_p^2 = .239$]. This was accounted for by significant group differences in discounting for small [$t(28) = 3.00$, $p = .006$;

Table 1
Mother and child demographics and drug-use summaries ($N = 60$)

	Smokers	Nonsmokers
Mother demographics		
Sex (n; female)	15	15
Age [years; M (S.D.)]	37.93 (6.18)	39.53 (5.89)
Race (n; white:black)	04:11	13:02**
IQ [M (S.D.)][a]	81.6 (13.81)	103.3 (11.79)**
Carbon monoxide [ppm; M (S.D.)]	21.13 (6.89)	2.67 (1.05)**
Cotinine [ng/ml; M (S.D.)]	2153.00 (875.47)	23.07 (29.97)**
Annual household income ($; Med)[b]	54279.00	59962.00
Mother drug use [M (S.D.)][c]		
Cigarettes	5.00 (0.00)	0.00 (0.00)**
Alcohol	2.47 (1.36)	1.87 (1.19)
Marijuana	0.53 (0.92)	0.00 (0.00)
Caffeine	3.57 (2.03)	3.87 (1.51)
Child demographics		
Sex (n; male:female)	9:6	6:9
Age [years; M (S.D.)]	12.53 (0.52)	12.53 (0.52)
IQ [M (S.D.)][a]	88.6 (16.83)	106.1 (15.53)**
Carbon monoxide [ppm; M (S.D.)]	2.80 (0.77)	2.47 (0.92)
Cotinine [ng/ml; M (S.D.)]	11.36 (31.03)	14.27 (24.91)
Child drug use [M (S.D.)][c]		
Cigarettes	0.27 (0.46)	0.00 (0.00)
Alcohol	0.27 (0.46)	0.27 (0.46)
Marijuana	0.13 (0.35)	0.00 (0.00)
Caffeine	1.47 (1.88)	3.07 (1.79)*

*$p < .05$, **$p < .01$ (two-tailed tests).

[a] IQ was assessed with the Kaufman Brief Intelligence Test, Second Edition (AGS Publishing).

[b] Annual household income is the median household income from 2007 adjusted census-tract data.

[c] Drug use was assessed with the following question: thinking about the past 6 months, how often have you used the following substances?: 0 = never tried, 1 = tried it, 2 = 1–2 times/month, 3 = once a week, 4 = 2–4 times/week, 5 = 5 or more times a week.

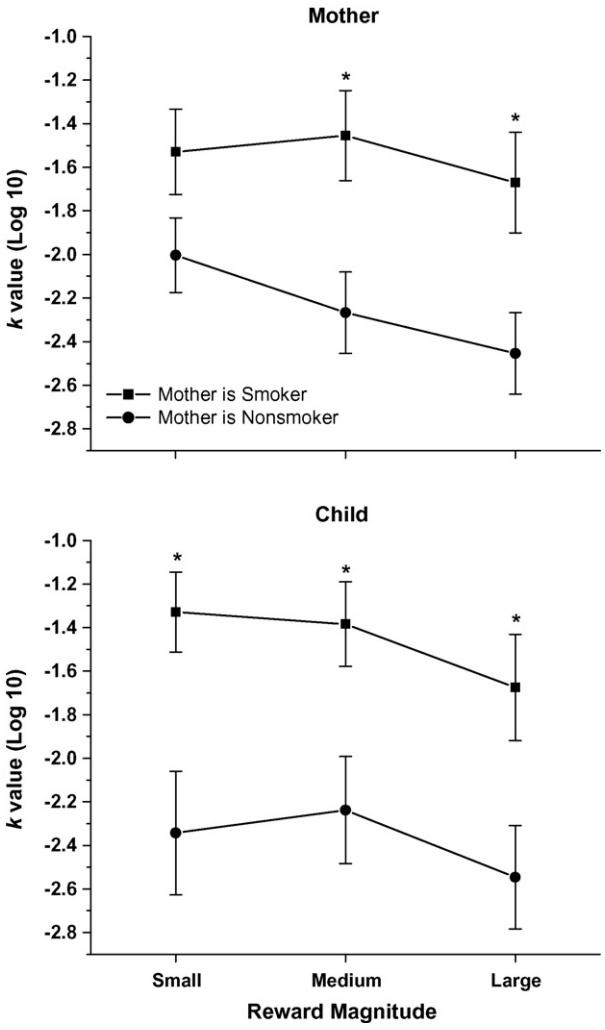

Fig. 1. Top panel: Group average Log 10 indifferences points (with S.E.M.) for small, medium, and large delayed rewards between mothers who smoke and do not smoke. Asterisk indicates significant group difference. Bottom panel: Group average Log 10 transformed indifference points (with S.E.M.) for children of mothers who smoke and do not smoke. Again, asterisk indicates significant group difference.

$\eta_p^2 = .243$], medium [$t(28) = 2.72$, $p = .011$; $\eta_p^2 = .209$], and large [$t(28) = 2.56$, $p = .016$; $\eta_p^2 = .189$] reward magnitudes (see bottom panel of Fig. 1). There was a modest but non-significant correlation for average k values between mothers and their children [$r(29) = .293$, $p = .123$; two-tail test].

Finally, group discounting differences based on mother smoking status were explored again while controlling for group differences in race and IQ (see Table 1). These controls reduced differences to non-significance ($ps > .05$) for mothers who did and did not smoke. However, group differences in discounting for their children remained significant ($ps < .05$) even after controlling for race and IQ.

4. Discussion

The current results are consistent with several earlier findings. These results confirm that adult smokes discount more by delay than nonsmokers (Bickel and Marsch, 2001; Reynolds, 2006). Also, children at high risk for cigarette smoking (i.e., mother is smoker) discounted significantly more by delay than children at lower risk of smoking. As such, delay discounting may represent a behavioral risk factor for cigarette smoking that predates any substantial use of nicotine. This would be consistent with findings based on other behavioral constructs (e.g., neurobehavioral disinhibition) that have been prospectively linked to drug use during adolescence and young adulthood (e.g., Kirisci et al., 2006). Similarly, high rates of delay discounting, or the tendency to *not* be behaviorally regulated by temporally distal events or consequences, may increase the likelihood that an individual will initially experiment with cigarettes and progress to more regular patterns of smoking. At these earliest stages of smoking, most of the associated negative consequences – which may serve to dissuade one from the initiation of smoking – are still in the future.

The current findings also were similar to findings reported in an earlier delay discounting study involving an at-risk sample (Petry et al., 2002). However, unlike the earlier research, we did not find gender differences in delay discounting among the at-risk youths—as reflected by the absence of interaction or main effects involving gender. It should be noted, though, that our sample was relatively small, and therefore there was limited power to detect interaction effects for gender. Otherwise, the current results are largely consistent with the earlier finding that individuals at risk for addiction discount more by delay than those at lower risk (Petry et al., 2002). But, the current study extends this earlier work by evaluating at-risk children (for cigarette smoking) instead of adults who were at risk for alcoholism but who had not become alcoholics. The next step in this research will be to prospectively explore possible links between delay discounting and the development of addictive behaviors. Additionally, future research will be needed to better understand the sources of these group differences between the children of smokers and nonsmokers. That is, it is still not known if these discounting differences are due to genetic factors, home environmental factors related to cigarette smoking, or perhaps smoking during pregnancy.

This study had limitations that should be addressed in future research. Again, the sample was small, which may limit generalization of the findings. Also, the small sample size may have constrained potential to identify a significant correlation between mothers and their children for delay discounting ($r = .29$, n.s.). Another limitation was not including fathers in the study. Some of the children of mothers who did not smoke may have had fathers who smoked, thus increasing their risk of smoking. However, this type of occurrence would increase similarity across the child comparison groups and presumably reduce the likelihood of group differences in delay discounting. Again, future research may explore these issues and more specifically isolate the relationship between delay discounting and the initiation of smoking.

Conflict of interest

All authors declare that they have no conflicts of interest.

Acknowledgements

We would like to thank Palak Shroff and Michele Patak for their expert assistance in conducting this research.

Role of funding source: This research was supported by an internal funding source at the Research Institute at Nationwide Children's Hospital. The Research Institute is a nonprofit organization and had no role in the planning and conduct of this research.

Contributors: The first author had primary responsibility for planning this research study and preparing the manuscript. The remaining three authors were responsible for data collection and help with analyses/manuscript preparation. All authors have reviewed and approved this submitted manuscript.

References

Bickel, W.K., Marsch, L., 2001. Toward a behavioral economic understanding of drug dependence: delay discounting processes. Addiction 96, 73–86.

Green, L., Myerson, J., 2004. A discounting framework for choice with delayed and probabilistic rewards. Psychol. Bull. 130, 769–792.

Jefferis, B., Graham, H., Manor, O., Power, C., 2003. Cigarette consumption and socio-economic circumstances in adolescence as predictors of adult smoking. Addiction 98, 1765–1772.

Kirby, K.N., Petry, N.M., Bickel, W.K., 1999. Heroin addicts have higher discount rates for delayed rewards than non-drug-using controls. Exp. Psychol.: Gen. 128, 78–87.

Kirisci, L., Tarter, R.E., Reynolds, M., Vanyukov, M., 2006. Individual differences in childhood neurobehavior disinhibition predict decision to desist substance use during adolescence and substance use disorder in young adulthood: a prospective study. Addict. Behav. 31, 686–696.

Maes, H.H., Neale, M.C., Kendler, K.S., Martin, N.G., Heath, C.A., Eaves, L.J., 2006. Genetic and cultural transmission of smoking initiation: an extended kinship model. Behav. Genet. 36, 795–808.

Petry, N.M., Kirby, K.N., Kranzler, H.R., 2002. Effects of gender and family history of alcohol dependence on a behavioral task of impulsivity in healthy subjects. J. Stud. Alcohol. 63, 83–90.

Reynolds, B., 2006. A review of delay-discounting research with humans: relations to drug use and gambling. Behav. Pharmacol. 17, 651–667.

Tyas, S.L., Pederson, L.L., 1998. Psychosocial factors related to adolescent smoking: a critical review of the literature. Tob. Control 7, 409–420.